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$0.00 0.076 DialUnits File410
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SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1966-2002/Jun W4
*File 155: Daily alerts are now available. This file has
been reloaded. Accession numbers have changed.
File 5:Biosis Previews(R) 1969-2002/Jun W3
(c) 2002 BIOSIS

Set Items Description
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? kfabp and insulin?
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>>>must use the FROM option to specify a file number.
? s kfabp and insulin?
3 KFABP
384383 INSULIN?
S1 2 KFABP AND INSULIN?
? rd
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S2 2 RD (unique items)
? t s2/3,ab/all

2/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10775878 20322410 PMID: 10866041
Adipocyte metabolism in adipocyte fatty acid binding protein knockout mice (aP2-/-) after short-term high-fat feeding: functional compensation by the keratinocyte [correction of keritinocyte] fatty acid binding protein.
Shaughnessy S; Smith E R; Kodukula S; Storch J; Fried S K
Department of Nutritional Sciences, Rutgers University, New Brunswick, New Jersey 08901-8525, USA.
Diabetes (UNITED STATES) Jun 2000, 49 (6) p904-11, ISSN 0012-1797 Journal Code: 0372763
Contract/Grant No.: DK38389; DK; NIDDK; F32 DK09303; DK; NIDDK
Erratum in Diabetes 2000 Sep;49(9) 1617
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Mice null for adipocyte fatty acid binding protein (AFABP) compensate by increasing expression of keratinocyte fatty acid binding protein (**KFABP**) (Hotamisligil et al. Science 274:1377-1379, 1996). In the present study, AFABP knockout (KO) and wild-type (WT) mice became equally obese on a high-fat diet, as judged by fat pad weights, adipocyte size, and body composition analysis. High-fat feeding led to moderate **insulin** resistance in both WT and AFABP knockout mice, as indicated by an approximately 2-fold increase in plasma **insulin**. However, in the high fat-fed mice, plasma glucose levels were approximately 15% lower in the AFABP-KO mice. Adipocytes isolated from AFABP-KO and WT mice fed high- or low-fat diets exhibited similar rates of basal and norepinephrine-stimulated lipolysis and **insulin**-stimulated rates of glucose conversion to fatty acids and glyceride-glycerol. However, basal glucose conversion to fatty acids was higher in adipocytes of AFABP-KO mice. Adipocyte tumor necrosis factor-alpha release was similarly increased by high-fat diet-induced obesity in both WT and AFABP-KO mice. As assessed by Western blot analysis, the level of **KFABP** protein in AFABP-KOs was

approximately 40% of the level of AFABP in WT controls. The binding affinities of **KFABP** for long-chain fatty acids were 2- to 4-fold higher than those of AFABP, but the relative affinities for different fatty acids were similar. As for AFABP, the rate of fatty acid transfer from **KFABP** to model phospholipid vesicles was increased with acceptor membrane concentration and by inclusion of acidic phospholipids, indicating a similar mechanism of transfer. We conclude **KFABP** can functionally compensate for the absence of AFABP, resulting in no major alterations in adipocyte metabolism or fat accumulation in response to short-term feeding of high-fat diets that result in moderate hyperinsulinemia.

2/3,AB/2 (Item 1 from file: 5)

13043726 BIOSIS NO.: 200100250875
Adipocyte metabolism in adipocyte fatty acid binding protein knockout (aP2-/-) mice after short-term high-fat feeding: Functional compensation by the keritinocyte fatty acid binding protein.
AUTHOR: Shaughnessy Sara; Smith Elizabeth R; Kodukula Sarala; Storch Judith (a); Fried Susan K(a)
AUTHOR ADDRESS: (a)Department of Nutritional Sciences, Rutgers University, 96 Lipman Dr., New Brunswick, NJ, 08901-8525: storch@aesop.rutgers.edu, sfried@rci.rutgers.edu**USA
JOURNAL: Diabetes 49 (6):p904-911 June, 2000
MEDIUM: print
ISSN: 0012-1797
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Mice null for adipocyte fatty acid binding protein (AFABP) compensate by increasing expression of keratinocyte fatty acid binding protein (**KFABP**) (Hotamisligil et al. Science 274:1377-1379, 1996). In the present study, AFABP knockout (KO) and wild-type (WT) mice became equally obese on a high-fat diet, as judged by fat pad weights, adipocyte size, and body composition analysis. High-fat feeding led to moderate **insulin** resistance in both WT and AFABP knockout mice, as indicated by an apprx2-fold increase in plasma **insulin**. However, in the high fat-fed mice, plasma glucose levels were apprx15% lower in the AFABP-KO mice. Adipocytes isolated from AFABP-KO and WT mice fed high- or low-fat diets exhibited similar rates of basal and norepinephrine-stimulated lipolysis and **insulin**-stimulated rates of glucose conversion to fatty acids and glyceride-glycerol. However, basal glucose conversion to fatty acids was higher in adipocytes of AFABP-KO mice. Adipocyte tumor necrosis factor-alpha release was similarly increased by high-fat diet-induced obesity in both WT and AFABP-KO mice. As assessed by Western blot analysis, the level of **KFABP** protein in AFABP-KOs was apprx40% of the level of AFABP in WT controls. The binding affinities of **KFABP** for long-chain fatty acids were 2- to 4-fold higher than those of AFABP, but the relative affinities for different fatty acids were similar. As for AFABP, the rate of fatty acid transfer from **KFABP** to model phospholipid vesicles was increased with acceptor membrane concentration and by inclusion of acidic phospholipids, indicating a similar mechanism of transfer. We conclude **KFABP** can functionally compensate for the absence of AFABP, resulting in no major alterations in adipocyte metabolism or fat accumulation in response to short-term feeding of high-fat diets that result in moderate hyperinsulinemia.

2000

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et     Items   Description
S1         0   MAL1 AND INSULIN?
S2         0   MAL (W) 1 AND INSULIN?
S3         8   KERATINOCYTE AND FATTY AND ACID AND BIND? AND INSULIN?
S4         5   RD (unique items)
? s afabp and insulin?
            26  AFABP
        384383  INSULIN?
      S5    8  AFABP AND INSULIN?
? s s5 not s4
             8  S5
             5  S4
      S6     6  S5 NOT S4
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...completed examining records
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 7/3,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13172398   21930042   PMID: 11932205
  Prolonged dietary treatment with conjugated linoleic acid stimulates
porcine muscle peroxisome proliferator activated receptor gamma and
glutamine-fructose aminotransferase gene expression in vivo.
  Meadus W J; MacInnis R; Dugan M E R
  Meat Research Section, Agriculture and Agri-Food Canada, Lacombe Research
Centre, 6000 C&E Trail, Alberta, Canada T4L 1W1. meadusj@em.agr.ca
  Journal of molecular endocrinology (England)   Apr 2002,  28  (2)  p79-86
,  ISSN 0952-5041   Journal Code: 8902617
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  Peroxisome proliferator activated receptors (PPARs) represent a family of
DNA binding proteins that are activated by a variety of dietary and
endogenous fatty acids. The PPAR proteins are expressed throughout the body
and are the target of a variety of lipidaemic and insulin sensitizing
drugs. Conjugated linoleic acid (CLA) is a collective name for
octadecadienoic acid isomers with conjugated double bonds, which can also
act as ligands for some of the PPAR family. To gain better understanding of
the long-term effects of PPAR activation, CLA was fed at 11 g/kg of feed
for 45 days to castrated male pigs (barrows). These barrows had a
significant repartitioning of subcutaneous fat to lean tissue in the
carcass: fat was reduced by 9 x 2% and lean muscle was increased by 3 x 5%,
but intramuscular fat content was also increased by 14% (P<0 x 05).
PPARgamma, glutamine-fructose aminotransferase (GFAT), adipocyte fatty acid
binding protein (AFABP ), but not PPARalpha mRNA levels were
significantly increased (P<0 x 05) in the CLA-fed pigs. The increased
expression of PPARgamma and AFABP indicates that CLA induced the
development of preadipocytes from stromal-vascular (s-v) stem cells to
promote intramuscular fat content. The increase in the expression of GFAT
mRNA indicates that the glucose supply of the muscle cells had been
increased with the CLA diet, possibly sparing intramuscular fatty acid
reserves.

 7/3,AB/2     (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08061704   94195269   PMID: 8145730
  Localization of a pioglitazone response element in the adipocyte fatty
acid-binding protein gene.
  Harris P K; Kletzien R F
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s Mal1 and insulin?
             95  MAL1
         384383  INSULIN?
      S1       0  MAL1 AND INSULIN?
? s mal (w) 1 and insulin?
          16372  MAL
        5522777  1
             43  MAL(W)1
         384383  INSULIN?
      S2       0  MAL (W) 1 AND INSULIN?
? s keratinocyte and fatty and acid and bind? and insulin?
          16381  KERATINOCYTE
         251460  FATTY
        2156501  ACID
        1197730  BIND?
         384383  INSULIN?
      S3       8  KERATINOCYTE AND FATTY AND ACID AND BIND? AND INSULIN?
? rd
...completed examining records
      S4       5  RD (unique items)
? t s4/3,ab/all

 4/3,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13009789   21884161   PMID: 11886494
  Human follicular papilla cells carry out nonadipose tissue production of
leptin.
  Iguchi M; Aiba S; Yoshino Y; Tagami H
  Department of Dermatology, Tohoku University School of Medicine, Sendai,
Japan.
  Journal of investigative dermatology (United States) Dec 2001, 117
 (6) p1349-56, ISSN 0022-202X Journal Code: 0426720
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  Leptin, a satiety-regulating cytokine, is predominantly expressed by
adipocytes, although recently the nonadipose tissue production of leptin
has been reported. To investigate the possibility of leptin production by
human scalp hair follicles, we examined leptin production and its mRNA
expression by cultured human follicular papilla cells. We isolated 12 human
follicular papilla cell lines from different individuals. They were
identified by their morphology, their high alpha-smooth-muscle actin
expression, their inability to differentiate into adipocytes, and by the
lack of mRNA for adipose-specific fatty acid binding
protein. All the human follicular papilla cell lines, but not neonatal
human dermal fibroblasts, produced significant amounts of leptin
demonstrable by enzyme-linked immunosorbent assay. We demonstrated leptin
mRNA expression by human follicular papilla cell lines, but not by neonatal
human dermal fibroblasts, by reverse transcription polymerase chain
reaction. By immunohistochemistry and in situ hybridization, we detected
both leptin protein and mRNA at the lower portion of the hair follicle,
i.e., hair matrix, inner root sheath of the hair bulb, and human follicular
papilla cells. In contrast, the leptin receptor with intracytoplasmic
signal sequence was detected in the follicular papilla cells
immunohistochemically, and the long isoform of the leptin receptor mRNA was
demonstrated in the human follicular papilla cell lines by reverse
transcription polymerase chain reaction. Finally, by using these human
follicular papilla cell lines, we showed that cytokines such as
interleukin-1 beta, tumor necrosis factor alpha, interferon-gamma, and
interleukin-4, and growth factors such as epidermal growth factor, basic
fibroblast growth factor, and transforming growth factor beta1, but not
vascular endothelial growth factor, hepatocyte growth factor,
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keratinocyte growth factor, and **insulin**-like growth factor 1, significantly downregulated the production of leptin. These data demonstrated that human follicular papilla cells produce leptin and express the functional leptin receptor in vivo and in vitro, suggesting its autocrine function. Moreover, the regulation pattern of its production by various factors suggests a pivotal role of leptin in hair biology.

4/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12748700 21549027 PMID: 11692175
Fatty acid binding protein expression in different adipose tissue depots from lean and obese individuals.
Fisher R M; Eriksson P; Hoffstedt J; Hotamisligil G S; Thorne A; Ryden M; Hamsten A; Arner P
Atherosclerosis Research Unit, King Gustaf V Research Institute, Karolinska Institute, Stockholm, Sweden. rachel.fisher@medks.ki.se
Diabetologia (Germany) Oct 2001, 44 (10) p1268-73, ISSN 0012-186X Journal Code: 0006777
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
AIMS/HYPOTHESIS: This study investigated the expression of adipose tissue **fatty acid binding** proteins (FABPs) in subcutaneous and visceral human adipose tissue depots from lean and obese individuals. METHODS: Adipocyte lipid **binding** protein (ALBP) and **keratinocyte** lipid **binding** protein (KLBP) expression was quantified by western blot in subcutaneous and omental adipose tissue from 20 obese and 9 lean individuals. RNA expression was quantified by Northern blot in the obese subjects. RESULTS: In the obese subjects, ALBP protein and RNA expression was higher in subcutaneous compared with omental adipose tissue (increases of 31 +/- 14 % and 40 +/- 13 % respectively, both $p < 0.05$), whereas in the lean group, KLBP protein levels were 32 +/- 9 % lower in subcutaneous fat ($p < 0.03$). However, the ALBP/KLBP ratio was greater in subcutaneous compared to omental adipose tissue from both lean and obese subjects: increases of 187 +/- 71 % ($p = 0.01$) and 52 +/- 23 % ($p = 0.17$) respectively for the protein ratio, and 21 +/- 6 % for RNA ($p = 0.01$, obese individuals). In lean subjects, **insulin** concentrations correlated positively with the ALBP/KLBP protein ratio in both depots (both $p <$ or $= 0.03$). CONCLUSION/INTERPRETATION: There are regional differences in adipose tissue FABP expression, which could be influenced by obesity. However, the ALBP/KLBP ratio is greater in subcutaneous than visceral adipose tissue in lean as well as in obese subjects. Investigation of adipose tissue FABPs could further our understanding of the role of **fatty** acids in the **insulin** resistance syndrome.

4/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10958186 20515662 PMID: 11060343
Lipid-**binding** proteins modulate ligand-dependent trans-activation by peroxisome proliferator-activated receptors and localize to the nucleus as well as the cytoplasm.
Helledie T; Antonius M; Sorensen R V; Hertzel A V; Bernlohr D A; Kolvraa S; Kristiansen K; Mandrup S
Department of Molecular Biology, University of Southern Denmark, Odense, DK-5230 Odense M, Denmark.
Journal of lipid research (UNITED STATES) Nov 2000, 41 (11) p1740-51, ISSN 0022-2275 Journal Code: 0376606
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: Completed

Peroxisome proliferator-activated receptors (PPARs) are activated by a variety of **fatty** acids, eicosanoids, and hypolipidemic and **insulin**-sensitizing drugs. Many of these compounds **bind** avidly to members of a family of small lipid-**binding** proteins, the **fatty acid-binding** proteins (FABPs). **Fatty** acids are activated to CoA esters, which **bind** with high affinity to the acyl-CoA-**binding** protein (ACBP). Thus, the availability of known and potential PPAR ligands may be regulated by lipid-**binding** proteins. In this report we show by transient transfection of CV-1 cells that coexpression of ACBP and adipocyte lipid-**binding** protein (ALBP) exerts a ligand- and PPAR subtype-specific attenuation of PPAR-mediated trans-activation, suggesting that lipid-**binding** proteins, when expressed at high levels, may function as negative regulators of PPAR activation by certain ligands. Expression of ACBP, ALBP, and **keratinocyte** lipid-**binding** protein (KLBP) is induced during adipocyte differentiation, a process during which PPARgamma plays a prominent role. We present evidence that endogenous ACBP, ALBP, and KLBP not only localize to the cytoplasm but also exhibit a prominent nuclear localization in 3T3-L1 adipocytes. In addition, forced expression of ACBP, ALBP, and KLBP in CV-1 cells resulted in a substantial accumulation of all three proteins in the nucleus. These results suggest that lipid-**binding** proteins, contrary to the general assumption, may exert their action in the nucleus as well as in the cytoplasm.

4/3,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10775878 20322410 PMID: 10866041

Adipocyte metabolism in adipocyte **fatty acid binding** protein knockout mice (aP2-/-) after short-term high-fat feeding: functional compensation by the **keratinocyte** [correction of keritinocyte] **fatty acid binding** protein.

Shaughnessy S; Smith E R; Kodukula S; Storch J; Fried S K
Department of Nutritional Sciences, Rutgers University, New Brunswick, New Jersey 08901-8525, USA.

Diabetes (UNITED STATES) Jun 2000, 49 (6) p904-11, ISSN 0012-1797
Journal Code: 0372763
Contract/Grant No.: DK38389; DK; NIDDK; F32 DK09303; DK; NIDDK
Erratum in Diabetes 2000 Sep;49(9) 1617
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Mice null for adipocyte **fatty acid binding** protein (AFABP) compensate by increasing expression of **keratinocyte fatty acid binding** protein (KFABP) (Hotamisligil et al. Science 274:1377-1379, 1996). In the present study, AFABP knockout (KO) and wild-type (WT) mice became equally obese on a high-fat diet, as judged by fat pad weights, adipocyte size, and body composition analysis. High-fat feeding led to moderate **insulin** resistance in both WT and AFABP knockout mice, as indicated by an approximately 2-fold increase in plasma **insulin** . However, in the high fat-fed mice, plasma glucose levels were approximately 15% lower in the AFABP-KO mice. Adipocytes isolated from AFABP-KO and WT mice fed high- or low-fat diets exhibited similar rates of basal and norepinephrine-stimulated lipolysis and **insulin**-stimulated rates of glucose conversion to **fatty** acids and glyceride-glycerol. However, basal glucose conversion to **fatty** acids was higher in adipocytes of AFABP-KO mice. Adipocyte tumor necrosis factor-alpha release was similarly increased by high-fat diet-induced obesity in both WT and AFABP-KO mice. As assessed by Western blot analysis, the level of KFABP protein in AFABP-KOs was approximately 40% of the level of AFABP in WT

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4/3,AB/5 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13043726 BIOSIS NO.: 200100250875
Adipocyte metabolism in adipocyte **fatty acid binding** protein knockout (aP2-/-) mice after short-term high-rat feeding: Functional compensation by the keritinocyte **fatty acid binding** protein.
AUTHOR: Shaughnessy Sara; Smith Elizabeth R; Kodukula Sarala; Storch Judith (a); Fried Susan K(a)
AUTHOR ADDRESS: (a)Department of Nutritional Sciences, Rutgers University, 96 Lipman Dr., New Brunswick, NJ, 08901-8525: storch@aesop.rutgers.edu, sfried@rci.rutgers.edu**USA
JOURNAL: Diabetes 49 (6):p904-911 June, 2000
MEDIUM: print
ISSN: 0012-1797
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Mice null for adipocyte **fatty acid binding** protein (AFABP) compensate by increasing expression of **keratinocyte fatty acid binding** protein (KFABP) (Hotamisligil et al. Science 274:1377-1379, 1996). In the present study, AFABP knockout (KO) and wild-type (WT) mice became equally obese on a high-fat diet, as judged by fat pad weights, adipocyte size, and body composition analysis. High-fat feeding led to moderate **insulin** resistance in both WT and AFABP knockout mice, as indicated by an apprx2-fold increase in plasma **insulin**. However, in the high fat-fed mice, plasma glucose levels were apprx15% lower in the AFABP-KO mice. Adipocytes isolated from AFABP-KO and WT mice fed high- or low-fat diets exhibited similar rates of basal and norepinephrine-stimulated lipolysis and **insulin** -stimulated rates of glucose conversion to **fatty** acids and glyceride-glycerol. However, basal glucose conversion to **fatty** acids was higher in adipocytes of AFABP-KO mice. Adipocyte tumor necrosis factor-alpha release was similarly increased by high-fat diet-induced obesity in both WT and AFABP-KO mice. As assessed by Western blot analysis, the level of KFABP protein in AFABP-KOs was apprx40% of the level of AFABP in WT controls. The **binding** affinities of KFABP for long-chain **fatty** acids were 2- to 4-fold higher than those of AFABP, but the relative affinities for different **fatty** acids were similar. As for AFABP, the rate of **fatty acid** transfer from KFABP to model phospholipid vesicles was increased with acceptor membrane concentration and by inclusion of acidic phospholipids, indicating a similar mechanism of transfer. We conclude KFABP can functionally compensate for the absence of AFABP, resulting in no major alterations adipocyte metabolism or fat accumulation in response to short-term feeding of high-fat diets that result in moderate hyperinsulinemia.

Upjohn Laboratories, Upjohn Company, Kalamazoo, Michigan 49001.
Molecular pharmacology (UNITED STATES) Mar 1994, 45 (3) p439-45,
ISSN 0026-895X Journal Code: 0035623
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

The thiazolidinediones are a class of antidiabetic compounds that increase the sensitivity of target tissues to **insulin**. An earlier study has shown that these compounds enhance the **insulin**-stimulated differentiation of 3T3-L1 cells and up-regulate expression of differentiation-dependent genes. We have observed that the mRNA encoding the adipocyte fatty acid-binding protein (**aFABP**) increases shortly after incubation of cells with pioglitazone, a thiazolidinedone analogue. The drug was found to enhance, in a time- and dose-dependent fashion, the expression of a chimeric gene that was constructed by fusing the **aFABP** promoter upstream of the chloramphenicol acetyltransferase (CAT) gene. To localize the sequence within the promoter that is responsive to pioglitazone, a series of chimeric genes containing sections of the **aFABP** promoter fused to the CAT gene were analyzed after transfection of 3T3-L1 cells. A section of DNA located at -5.2 kilobases and known to encompass a tissue-specific and differentiation-dependent enhancer element was found to confer responsiveness to the drug. Analysis of sequences in this region of the **aFABP** promoter by DNA gel retardation assays revealed the presence of a protein in nuclear extracts from drug-treated cells that bound to a specific sequence (ARE-6). The presence of the protein could be demonstrated in differentiated adipocytes, but the protein was present at only low levels in preadipocytes. Treatment of preadipocytes with pioglitazone resulted in the precocious appearance of this protein in nuclear extracts. Multiple copies of the ARE-6 sequence inserted upstream of a heterologous promoter linked to the CAT gene conferred pioglitazone responsiveness. The experiments reported in this study establish that the **insulin**-sensitizing agent pioglitazone up-regulates expression of the **aFABP** gene through an element located within a region of DNA responsible for tissue-specific and differentiation-dependent expression of the gene.

7/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07537653 93062815 PMID: 1435736

Adipocyte fatty acid-binding protein: regulation of gene expression in vivo and in vitro by an **insulin**-sensitizing agent.

Kletzien R F; Foellmi L A; Harris P K; Wyse B M; Clarke S D

Metabolic Diseases Research, Upjohn Laboratories, Upjohn Company, Kalamazoo, Michigan 49001.
Molecular pharmacology (UNITED STATES) Oct 1992, 42 (4) p558-62,
ISSN 0026-895X Journal Code: 0035623
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Pioglitazone, a thiazolidinedione, is a novel antidiabetic compound that can lower blood glucose in diabetic rodents by increasing **insulin** sensitivity in target tissues. We have previously demonstrated that pioglitazone can enhance the **insulin**- or **insulin**-like growth factor-1-regulated differentiation of 3T3-L1 cells, a cell line that undergoes morphological and biochemical differentiation to mature adipocytes [Mol. Pharmacol. 41:393-398 (1992)]. In this study, we have examined the effect of pioglitazone on the expression of the adipocyte fatty acid-binding protein (**aFABP**) in ob/ob mice and 3T3-L1 cells. Administration of the drug to mice was observed to cause a dose-dependent increase in **aFABP** mRNA expression in epididymal fat, which was

correlated with a decrease in blood glucose and **insulin** levels. Treatment of 3T3-L1 cells with pioglitazone enhanced **aFABP** expression in a time-dependent fashion. To explore a possible direct effect of pioglitazone on **aFABP** expression, a chimeric gene was constructed containing the **aFABP** promoter fused upstream of the bacterial reporter gene for chloramphenicol acetyltransferase. After transfection into 3T3-L1 cells and selection of stable transformants, regulation of the chimeric gene was studied. Pioglitazone, in combination with **insulin** or **insulin** -like growth factor-1, was observed to elicit a dose-dependent increase in expression, indicating a role for pioglitazone in regulating transcription of the **aFABP** gene. Several thiazolidinedione analogs were tested for their ability to induce the expression of the chimeric gene, and it was found that activity in this assay paralleled the structure-activity relationships observed for enhancement of 3T3-L1 cell differentiation. These observations on control of **aFABP** gene expression by pioglitazone suggest possible mechanisms by which cellular sensitivity to **insulin** may be regulated.